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A CASE OF ACUTE SPINAL PARALYSIS; DEATH ON THE TWELFTH DAY; AUTOPSY SHOWING TRANSVERSE CERVICAL MYELITIS.[1]

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THE following case is of interest because, while having the clinical history both of acute poliomyelitis anterior and of Landry's paralysis, the post-mortem results show a transverse myelitis involving only the cervical portion of the cord:

The patient was a well-grown young man, eighteen years of age, of good habits, and without hereditary history of consumption or nervous disease. He was an only child, and had enjoyed excellent health until July, 1890. On the 10th of this month he went to New York for the day, and in the afternoon went to Coney Island. During the day the weather was intensely hot, but by evening there was a great change in the temperature, the thermometer falling several degrees. He suffered extremely from the cold, feeling chilled through. The next day he returned home to Philadelphia, and felt very weak. The day following he was a little

[1] Read at the meeting of the American Neurological Association held in Washington, D. C., September 22 to September 25, 1891.

feverish, and complained of general weakness and relaxation, with a sense of general numbness, especially from the hips down. He tottered a little in his walk, and had some cramp-like pains in the calves of his legs. He had a little pain in his back and head, and was drowsy and dull. There was some diarrhea. There was pain on movement, and when sitting, his legs seemed as if "asleep." The next day, July 12th, he was worse; he could not walk, his legs being too weak. In the evening of this day he found that there was some loss of power in his arms. This loss of power increased steadily, and by July 14th he was unable to move his legs at all, though still able to lift the arms and move the hands and fingers. There was temporary inability to void urine, and a little difficulty in swallowing. The bowels were moved by enemata.

On July 18th I saw the patient in consultation with Dr. Radcliffe Cheston, who gave me the foregoing history. The patient had been confined to bed for a week, and was steadily losing strength. I made the following notes of his condition: "There is slight paralysis of the left side of the face. He cannot whistle, and smiling draws the face to the right. The eyesight is good, although on the first day of his illness vision of the right eye was somewhat blurred. He can turn the head to either side; can extend it, but cannot flex it. He cannot extend either arm, but can partially adduct the arms over the chest. He can flex and extend both forearms, can pronate, but cannot supinate them; can flex, but can only partially extend the fingers. He can feebly extend te last phalanges, and can flex and extend the wrists. The grip is very feeble. He cannot raise the body or turn it to either side, owing to paralysis of the muscles of the trunk. There is complete loss of power in both legs. He cannot

move a muscle, except to a slight extent the flexors and extensors of the toes. There is loss of most reflexes. The knee-jerk is entirely absent; no plantar or abdominal reflex is present, but the cremasteric and epigastric reflexes can be excited.

"Sensation is unimpaired in the upper and lower extremities, except as to compass-points. There is slight loss to these on the fingers and on the feet, the points being distinguished only when far apart. He locates well, and the pain-sense is unaffected. He says that the hands and feet feel numb and tingling, but he has no pain anywhere; on the day before, however, he had some cramp in his legs. There is no muscular tenderness or pain on pressure over the nerve-trunks. All the muscles respond to a slowly interrupted faradic current. The thigh muscles do not react as readily to the current as the other muscles. Speech is unaffected, but his voice is weak. He is much annoyed by accumulation of mucus in his throat."

On July 22d the patient had a return of difficulty in swallowing, and once or twice he had to be fed through the nasal tube. The respirations had become rapid and shallow. There was retention of urine the previous day, and the catheter had to be used, but he urinated to-day without trouble. The patient gradually lost strength, and died of respiratory paralysis July 22d, twelve days after the onset of the attack. The temperature during the attack was from 100° to 101°. The post-mortem was made forty-eight hours after death, the body having been preserved in an ice-box.

Report of Autopsy by Dr. C. W. Burr.

The body was of average size and weight; the abomen was of a slightly greenish color. The scalp, the skullcap, and the dura mater were normal.

The brain was of average size, and there was no edema or congestion. The convolutions showed no marked deviations from the common type. On cutting into the brain the gray and white matter showed well, and were of good consistency. The basal ganglia, pons, and medulla were healthy. The sciatic nerves were also healthy. The bones of the base of the skull and of the spinal canal showed no evidence of disease.

The spinal meninges were normal, and there was no excess of spinal fluid. The cord was of good color, form, and consistency throughout, except for apparent slight softening in the lower dorsal region, due probably to post-mortem change. On cross-section the gray and white matter were throughout well differentiated, as well in the dorsal as in the other regions. There was possibly some congestion at the cervical swelling.

After hardening for three months in Müller's fluid the following conditions were found: The cervical swelling still showed well the differentiation between white and gray matter, but it was crumbly and brittle, and irregular longitudinal fissures appeared in several places, notably in the posterior half (these occurred during hardening). It was misshapen in outline. The staining was normal throughout. The remainder of the cord, brain, etc., showed nothing abnormal.

Microscopical Examination.

Pieces were imbedded in celloidin and sections stained with carminate of soda and with Congo-red, and a few by Pal's method.

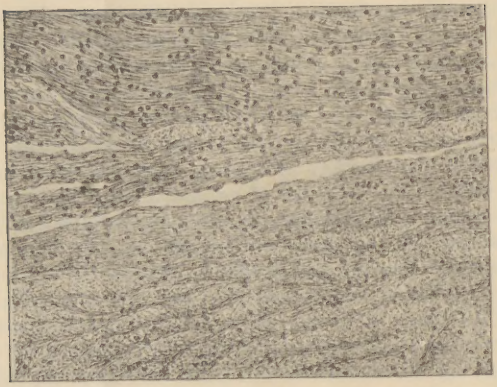
Cervical swelling. There was marked infiltration by small, round, sometimes irregularly shaped, deeply stained cells, which invaded the entire transverse section of the cord. Their distribution was not uniform, and the position of greatest in-

tensity varied with the level of the section. In the center of the anterior gray matter on one side and posteriorly on the other were found small areas of hemorrhage and softening. There was some engorgement of capillaries. A few of the motor cells were shrunken and without processes, but most of them were normal in every respect—in form, intensity of staining, processes, appearance of protoplasm, and character of nuclei. Here and there, in irregularly-distributed spots, the nerve fibers had disappeared, but quite the larger number were still present. The pia and the posterior nerve-roots, especially the latter, were involved in the cellular infiltration. The vertical extent of the lesion was about one inch—sections from other portions of the cord, from the medulla and pons, and from the brain and sciatic nerves, showing nothing pathological.

Pathologically, the case is one of acute transverse myelitis, involving the upper part of the cervical swelling. The palsy of respiration may have been due to chemical changes sufficient to inhibit function, but not sufficient to cause material alteration of structure. The abdominal and thoracic viscera were not examined.

The accompanying illustrations made after drawings by Dr. Allen J. Smith clearly show the appearances described.

From a study of the clinical features of the case the diagnosis lies between acute multiple neuritis, acute myelitis, Landry's paralysis, and polio-myelitis anterior. Multiple neuritis can be excluded because of the absence of pain in the nerve trunks, the absence of hyperesthesia, the fact that sensibility was so little disturbed, the early loss of the reflexes, and the rapid course of the disease. Acute myelitis is



From posterior root zone.

Anterior horn, showing cellular infiltration.

accompanied with early loss of power in the sphincters, exaggerated reflexes if the disease is above the lumbar enlargement, pain in the back, contraction of the limbs, and trophic changes in the skin—symptoms all absent in this case. The symptomatology of Landry's paralysis closely resembles that of my patient, but I believe that in the typical form of acute ascending paralysis the loss of power begins in the lower extremities and spreads systematically upward. In my case, although the paralysis did begin in the legs, the loss of power in the rest of the voluntary muscles came on almost simultaneously. Still, I admit that without a post-mortem examination it would have been next to impossible to say that the case was not one of Landry's paralysis. Dr. Henry Hun read before the New York Neurological Society a valuable paper on the pathology of acute ascending paralysis.[1] The case reported by him is so strikingly like the one I have related that I will give it at some length.

An unmarried man of forty-five years, free from any syphilitic or rheumatic taint, was suddenly attacked, April 11, 1890, by a paresis of the legs, which in the space of four days became a complete paralysis, and then in a few days more there was loss of power in the arms and hands. He had a difficulty in speech. It was necessary to draw the urine with a catheter. The bowels were constipated. This seemed due rather to weakness of the abdominal muscles than to paralysis of the bladder and rectum. He was admitted to the Albany Hospital April 18, 1890. Ten days after the beginning of weakness in

[1] Journal of Nervous and Mental Disease, June, 1891.

the legs Dr. Hun made the following note of the patient's condition: "Well nourished. Ptosis of right eyelid, which he says is voluntary and due to impairment of vision. This impairment of vision seems to consist of diplopia, one object being to the right of and lower than the other. Decided paralysis of lower branch of left facial nerve. Tongue protruded straight, no tremor of tongue or lips, and no muscular atrophy or fibrillary contraction. Breath very offensive and tongue covered with a white coat. He can whistle with difficulty. Speech thick and indistinct. Deglutition so difficult that he has to be fed with stomach tube. Mucus collects in trachea and patient can eject it very imperfectly. Hearing, smell, and taste are normal. Grasp of left hand *nil*. Grasp of right hand very feeble. Flexors and extensors of upper arm very feeble, but more powerful than those of forearm. Muscles of shoulder feeble, but more powerful than those of arm. Muscles of body paralyzed so that he can neither sit up nor move in bed. Absolute motor paralysis of both legs and thighs. Absence of plantar, cremasteric, umbilical, and patellar reflexes. No muscular tenderness, fibrillary contraction, or atrophy. No disturbance of sensibility, tactile (tested with pin-head and cotton), thermic, or painful impressions anywhere. No retardation of conduction of pain. Bladder and rectum inactive. Urine drawn off with catheter. Bowels moved by castor oil or by injection. Temperature and pulse normal. On April 22d, the muscles of both arms are much weaker, and those of the right arm and shoulder are weaker than those of the left, so that he can scarcely move the right arm at all. The muscles of the legs continue completely paralyzed. There is nowhere any muscular atrophy, tremor, or disturbance of any kind of sensibility, and there is no muscular tenderness. All

the muscles of both legs respond decidedly and quickly to the faradic current. The difficulty of speech and respiration is so great that it does not seem possible that he can live twenty-four hours."

The patient died April 23d, five days after admission to the hospital, and twelve days after the beginning of loss of power in the legs.

"An autopsy was held eight hours after death. The spinal cord seemed normal, except that the lumbar portion seemed to be slightly edematous, and the outlines of the gray matter in this region seemed a little less clear than normal. The brain seemed normal, except that there was a somewhat increased amount of subarachnoid fluid over both parietal regions."

"A microscopical examination made by Dr. Van Gieson showed that there were a slight cerebral and spinal meningitis and infiltration of the walls of some of the veins of the spinal pia mater, and a degeneration (or neuritis) of some of the fibers of the anterior roots of the cauda equina, the nervous system in other respects being normal."

R. T. Williamson, M.D., has reported a case of acute anterior polio-myelitis in a young man, twenty-two years old, who died five weeks after the onset of the attack.[1] The case is as follows:

The patient, aged twenty-two years, consulted Dr. Pullon. When first seen he complained of a numb feeling in the right hand. The next day this had extended to the right leg and also to the left side. The right arm became paralyzed, then the left, and on the third day there was complete paralysis of both arms and legs. There was also pain in the back and slight pain in the limbs. The knee-

[1] Medical Chronicle, September, 1890, page 454.

jerks were absent; there was no anesthesia anywhere. The bladder and rectum were not affected. Rapid atrophy of the muscles of the limbs occurred after the first ten days of the illness. He remained in this paralyzed condition for about three weeks, then he began to improve. He regained slight power of movement in the toes, and there was slight return of power of movement in both arms. For financial reasons he was admitted as a patient at the Victoria Hospital, Burnley, England. The day following his admission he died very suddenly, before the hospital doctor could be called to see him. The entire duration of the disease was about five weeks. There was no anesthesia during the entire illness, and no affection of the bladder and rectum. The post-mortem examination was made by Dr. Woodyatt, the house-surgeon of the Burnley Hospital.

The spinal cord before section was normal in appearance, and so were the meninges. The cause of the sudden death was not discovered. Portions of the cervical, dorsal, and lumbar regions of the spinal cord, and pieces of the sciatic and ulnar nerves, were placed in bichromate of potash solution and sent to me for microscopical examination. To the naked eye the cord on section appeared normal, with the exception of the anterior horns of gray matter, which were of a slightly lighter color than the rest of the gray matter. The specimens were hardened in bichromate of potash solution and imbedded in celloidin. Transverse sections were cut and stained with logwood, anilin blue-black, osmic acid, Weigert's stain, etc. On microscopical examination of the spinal cord changes were found in each anterior horn of the gray matter in all three regions, cervical, dorsal, and lumbar, the most marked being those in the lumbar region, those in the dorsal, however, but slight. The changes occupied

almost exactly the same position in all three regions, viz., the outer part of each anterior horn. The rest of the gray matter was not affected. In a transverse section of the lumbar region, stained with logwood, the outer half of each anterior horn of the gray matter was seen to be infiltrated by a mass of closely packed round cells. The cell-nuclei stained well with logwood. Under a low power the stained cell-nuclei were seen to be more numerous around the periphery of the patch than at the center. Also the center of the cell-infiltration had a tendency to break up and fall out of the sections.

A line drawn transversely across the cord through the central canal would roughly limit the post-anterior part of the patch of cell-infiltration, and a line drawn in the antero-posterior direction through the center of the anterior horn would give roughly the inner boundary of the patch. When examined with a high power this roundish patch of cell-infiltration was seen to be composed of small, round, nucleated cells, about the size of white blood-corpuscles, and of large, round, or oval nucleated cells. There were no real hemorrhages; only here and there two or three stray red corpuscles were seen, apparently not surrounded by any capillary wall. The perivascular sheaths of all the vessels were greatly distended with round cells. The small arteries passing from the surface of the cord to the anterior horns were dilated, and the lymph sheaths distended with round cells. This was especially the case with the lateral median and lateral anterior arteries and their branches.

Nerve-cells. In the lumbar region, in specimens stained according to Weigert's method or with anilin blue-black, no nerve-cells could be detected in the outer half of each anterior horn—the region of the round-cell infiltration. This corresponded to the

region of the antero-lateral and postero-lateral, and the central group of ganglion cells. At the inner part of each anterior horn—the region of the anterior, the internal, and the median groups—ganglion cells were seen. Some were of normal appearance, but most, especially those in the immediate neighborhood of the patch of round-cell infiltration, were shrunken and had lost their processes.

Anterior nerve-roots. Transverse section. In each nerve-bundle only a few nerve fibers were seen (stained black by Weigert's method), and instead of lying close together as in health they were widely separated from each other. Between these fibers was an infiltration of small round cells, well marked in some bundles of fibers, in some only slight; in others it was absent. Between the fibers, also, were many clear, unstained, more or less circular spaces, from which the nerve-fibers had entirely disappeared. At some places the nerve-fibers were represented by large masses of myelin (stained black).

Pieces of the anterior nerve-roots, teased out, showed the white substance of Schwann broken up into irregularly-shaped masses, or into round or oval globules. The nerve-nuclei were greatly increased in number. At some spots both white substance and axis cylinder had entirely disappeared and only the external sheath was left. The posterior nerve-roots on section presented a marked contrast to the anterior nerve-roots, appearing almost normal.

In the cervical region of the cord the changes were much the same and occurred in the same position, *i. e.*, the external part of the anterior horn; they were not so well marked, however, as in the lumbar region.

In other points the description given of the lumbar region applies to the cervical region. The pia mater was normal with the exception of very slight

cell-infiltration and dilatation of the vessels of the anterior part just at the region of the anterior nerve-root.

Filum terminale. In the anterior horns the vessels were dilated. The perivascular sheaths of some of the dilated vessels contained a considerable number of round cells. This was most marked at the outer part of each horn. The nerve-cells were normal, except that at the outer part of the anterior horn one or two nerve-cells in each section could be detected, the processes of which were not quite so distinct as in health. In all four regions of the cord the changes were most marked just at the spot where the antero-lateral artery entered the gray matter, and the perivascular sheath of this artery was nearly always distended with round cells. Sections of the cord were stained for microörganisms according to Weigert's modification of Gram's method, but none were detected.

The ulnar nerve just above the wrist, both on section and in a teased specimen, showed many degenerated fibers similar to those of the anterior root. Other fibers, however, were normal.

The sciatic nerve. On transverse section many fibers had a normal appearance, but in each bundle here and there were small spaces from which the nerve-fibers had disappeared. A piece of the nerve, teased, showed some degenerated fibers. Others were normal. The smaller branches of the peripheral nerves and the medulla were not preserved for microscopical examination.

The clinical histories of the three cases detailed are in most respects alike; except that in Dr. Williamson's case the course of the disease was much longer, and there had been an attempt at retrogression of the paralysis, and there was also marked

atrophy of the muscles. The post-mortem findings were all different. In Dr. Hun's case no changes could be found in the cord; in my own there was myelitis of the cervical portion of the cord, and in Dr. Williamson's there was marked degeneration of the anterior horns and some changes in the nerve-cells. It seems to me probable that had my patient lived long enough there would have been some retrogression of the paralysis, and changes in the muscles, both as to atrophy and in the electrical reactions, would have taken place.

I am inclined to believe that the condition of the cord found in my case was due to the early stage of the disease at which the patient died. Had he lived as long as did Dr. Williamson's patient, it is possible there would have been less myelitis, but degeneration in the anterior horns would have been conspicuous. I think it probable that in the early stages of poliomyelitis anterior there is a hyperemia or inflammation of the cord in the affected areas that not confined to the anterior horns. After a few days, if the course of the disease tends toward recovery, the general myelitis disappears and the degenerative changes in the anterior horns become pronounced. The large ganglion cells become the seat of special changes; they lose their processes, shrivel, and many disappear altogether. This seems likely from the clinical history of a case of this disease. There are at first associated with the motor paralysis certain subjective sensory disturbances in the way of formication, numbness, pain in the back and limbs, and discomfort on movement. In a few days the sensory phenomena disappear—in fact they seldom

last more than a week; soon after the relief of the sensory symptoms retrogression of the paralysis takes place in those limbs that are destined to recover completely.

There have been very few autopsies in the early stages of polio-myelitis anterior, and some of those that have been reported will not bear close inspection For instance, the case reported by Charlewood Turner,[1] which is quoted by Gowers, Mary Putnam Jacobi, and others, does not seem to me to have been a case of polio-myelitis anterior:

The patient was a child two and one-half years of age. She had slipped in going down stairs, and had fallen on her back. For two weeks after this she had played about as usual. She then, one day, complained of being cold, became quite ill, and on the same day lost power in her legs. Three days later she could not use the arms. On admission to the London Hospital she was suffering from paralysis of all her limbs. She was anemic; there was fairly good muscular development. She lay on her side with her head thrown backward and the legs extended. The head could be rotated and bent backward, but could not be flexed without pain. There was no reflex action on tickling the soles of the feet, and *sensation over the legs was lost.* Sensation in the arms was perfect. The evacuations were passed involuntarily. Twelve days later the evacuations were still involuntary, but sensation was restored to the legs. The child died twenty-six days after admission and six weeks after the onset of the paralysis. At the autopsy there was found in the lumbar region of the cord hemorrhagic softening in the anterior horns, leaving a cavity.

[1] Trans. Pathological Society of London, vol. xxx. p. 202.

Drummond's[1] case, the autopsy of which is the earliest on record in a case of polio-myelitis anterior, is incomplete, from the absence of clinical history:

A child, five years of age, died after an illness of six or seven hours, from respiratory paralysis. There is no mention made of loss of power in the legs or arms. At the post-mortem examination "the spinal cord in the region of the fourth and fifth cervical nerves showed undue redness in the anterior gray matter. The vessels running from the surface to the cornua were distended with blood. The microscope showed distention of capillaries and minute extravasations in the gray substance, swelling of the neuroglial elements and of the ganglion cells, which were granular with indistinct processes. The blood-vessels were distended with blood, forming a striking feature, not only in the anterior horns, but in the anterior white columns and middle and anterior portions of the posterior columns."

Drummond lays stress upon the fact that only a limited portion of the cord was affected, that is, in the region of the fourth and fifth cervical nerves, and that the inflammation extended beyond the limits of the anterior horns. The changes were found most abundantly in the anterior cornua, but they were also found in the antero-lateral white columns and in part of the posterior horns. "In fact, the anterior cornua were only the centers of inflamed areas, which reached considerably beyond their limits. It will therefore become a question whether the name proposed by Kussmaul, and adopted by the majority of writers, anterior poliomyelitis, is really an appropriate one."

[1] Brain, 1885, p. 15.

Angel Money's[1] case also showed that the inflammation was not confined to the anterior horns:

The patient was a child, two years of age, that died sixteen weeks after the onset of an attack of paralysis involving both legs. The autopsy "showed changes in the lumbar enlargement, distention and thrombosis of the vessels, especially in the anterior horns. The cornua were infiltrated with leucocytes. There was absence of the large multipolar and other nerve-cells. The disease was not confined to the anterior horns, but spread forward, outward, and backward. The principal focus of mischief was certainly the center of the anterior horns."

Gowers has recently reported a case[2] of acute polio-myelitis associated with multiple neuritis. He refers to the two varieties of neuritis that are met with, the parenchymatous and the adventitious, using the latter term to designate the variety in which the connective-tissue elements were primarily affected. "Recent research," he says, "has suggested that there are two varieties of polio-myelitis, the primary affection in one being in the nerve-cells of the anterior horns, in the other in the basic substance of gray matter."

There has been an effort made of late to attribute Landry's paralysis to acute multiple neuritis, but the cases that have been cited present the features of multiple neuritis and not those of acute ascending paralysis.

[1] Trans. Pathological Society of London, 1884.

[2] Clinical Society of London, and American Journ. Med. Sci. June, 1891.

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